Results of surgery for irreversible moderate to severe mitral valve regurgitation secondary to myocardial infarction

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Abstract: Objective: Moderate to severe irreversible mitral regurgitation secondary to myocardial infarction is an independent risk factor for reduced long-term survival. Late effects of correction of mitral incompetence concomitant with coronary artery bypass grafting (CABG) are less well known and the choice of mitral valve procedure is still debated. Methods: From 1988 to 1998, 93 consecutive patients (mean age 63±9 years) were treated for moderate to severe irreversible mitral regurgitation secondary to myocardial infarction; 84 were in NYHA functional class III-IV and 19 were in cardiogenic shock. Thirty-seven patients underwent emergency surgery. Perioperative intraaortic balloon pump (IABP) was necessary in 33 patients. Follow-up ranged from 6 months to 12 years (mean 51 months±41). Results: Mitral valve was repaired in 30 patients and replaced in 63. Replacement was preferably performed in patients with major displacement of papillary muscle and in patients with acute papillary muscle rupture. CABG (3.4 distal anastomoses) was performed in all patients and was complete in 92%. Early mortality was 15% (14/93). Multivariable analysis identified need for IABP (P=0.005) and COPD (P=0.02) as risk factors for early death. Emergency surgery had only a trend (P=0.15) for increased mortality; age, low ejection fraction, repair vs. replacement had no influence. Actuarial survival rates at 1, 5 and 10 years were 81, 65 and 56%, respectively. Late survival was similar in patients with replacement or repair (P=0.46). At last follow-up, all but one patient were in NYHA functional class I or II. Conclusions: Combined mitral valve procedure and myocardial revascularization, as complete as possible, for moderate to severe mitral regurgitation secondary to myocardial infarction achieve satisfactory early and late outcome despite the increased operative mortality. Acute papillary muscle rupture, severe restriction of the mitral valve by major displacement of the papillary muscle are better managed by valve replacement

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Results of surgery for irreversible moderate to severe mitral valve regurgitation secondary to myocardial infarction ☆

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Abstract

Objective: Moderate to severe irreversible mitral regurgitation secondary to myocardial infarction is an independent risk factor for reduced long-term survival. Late effects of correction of mitral incompetence concomitant with coronary artery bypass grafting (CABG) are less well known and the choice of mitral valve procedure is still debated. **Methods**: From 1988 to 1998, 93 consecutive patients (mean age 63 ± 9 years) were treated for moderate to severe irreversible mitral regurgitation secondary to myocardial infarction; 84 were in NYHA functional class III–IV and 19 were in cardiogenic shock. Thirty-seven patients underwent emergency surgery. Perioperative intraaortic balloon pump (IABP) was necessary in 33 patients. Follow-up ranged from 6 months to 12 years (mean 51 months ± 41). **Results**: Mitral valve was repaired in 30 patients and replaced in 63. Replacement was preferably performed in patients with major displacement of papillary muscle and in patients with acute papillary muscle rupture. CABG (3.4 distal anastomoses) was performed in all patients and was complete in 92%. Early mortality was 15% (14/93). Multivariable analysis identified need for IABP ($P = 0.005$) and COPD ($P = 0.02$) as risk factors for early death. Emergency surgery had only a trend ($P = 0.15$) for increased mortality; age, low ejection fraction, repair vs. replacement had no influence. Actuarial survival rates at 1, 5 and 10 years were 81, 65 and 56%, respectively. Late survival was similar in patients with replacement or repair ($P = 0.46$). At last follow-up, all but one patient were in NYHA functional class I or II. **Conclusions**: Combined mitral valve procedure and myocardial revascularization, as complete as possible, for moderate to severe mitral regurgitation secondary to myocardial infarction achieve satisfactory early and late outcome despite the increased operative mortality. Acute papillary muscle rupture, severe restriction of the mitral valve by major displacement of the papillary muscle are better managed by valve replacement.

Keywords: Mitral regurgitation; Myocardial infarction

1. Introduction

Mitral regurgitation secondary to myocardial infarction is a common clinical problem, developing after myocardial infarction in up to 19% of patients [1]. Uncorrected moderate to severe mitral regurgitation secondary to myocardial infarction during coronary artery bypass grafting (CABG) is an independent risk factor for long-term survival [1–3]. Catheter-based reperfusion with thrombolysis or coronary angioplasty does not reliably reverse valvular incompetence nor does it reduce mortality to the levels experienced by patients with lesser degrees of mitral regurgitation [4]. Thus, an increasing number of patients with moderate to severe mitral regurgitation secondary to myocardial infarction require combined surgical procedures to revascularize the ischemic myocardium and to restore mitral valve competence. However, late effects of correction of mitral incompetence concomitant with CABG are less well known [5–8] and the choice of mitral valve procedure is still debated. In this study, we report the results of combined mitral valve and coronary artery bypass procedures, and our indications for the choice of mitral valve procedure in specific subsets of these patients.

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2. Patients and methods

2.1. Definitions

Mitral valve regurgitation secondary to myocardial infarction was documented angiographically and/or echocardiographically and graded by taking into account several echocardiographic criteria [7,9–11]:

- the proximal jet diameter measured in the cross-section in which the minimal diameter of the mitral regurgitation on the atrial side appeared the largest, a diameter of >6.5 mm relates to mitral regurgitation grades 3 and 4. The radius of the proximal isovelocity hemicircle with a Nyquist limit set at 35–40 cm/s helps to differentiate between grade 2 (0–3 mm), grade 3 (3–9 mm) and grade 4 (>9 mm);
- the color Doppler jet area: 0–1.5 cm^2 for grade 1, 1.5–3 cm^2 for grade 2, 3–6 cm^2 for grade 3, >6 cm^2 for grade 4;
- the intensity of the continuous flow Doppler. The density and completeness of a continuous wave signal from a regurgitant jet is a function of the angle at which the jet is interrogated and the number of reflecting red cells flowing through the laminar core. In grade 4 mitral regurgitation, if the proper interrogation angle is obtainable, the regurgitant jet is dense and has a sharp border or envelope;
- the systolic Doppler flow in the pulmonary veins: the pulmonary flow is normally maximal during early systole. Trivial grade 1 mitral regurgitation does not alter the pulmonary vein flow, but as mitral regurgitation reaches moderate and severe degrees, systolic inflow becomes progressively blunted. In severe grade 4 mitral regurgitation the flow reverses; it is biphasic in moderate grade 3 mitral regurgitation and only diminished in mild grade 2 mitral regurgitation.

In this study, indication for mitral valve surgery was grade 3 or grade 4 irreversible mitral regurgitation secondary to myocardial infarction. All five patients who underwent combined mitral valve and revascularization procedures because of inability to discontinue the cardiopulmonary bypass after isolated revascularization, had grade 2 mitral regurgitation preoperatively. Otherwise, this study does not deal with grade 2 mitral regurgitation secondary to myocardial infarction.

Coronary stenosis was considered significant if there was a 70% or greater stenosis in the luminal diameter in any view. A stenosis of 50% or more in the left main coronary artery was considered significant [3].

Patients were excluded if there was evidence of other mitral valve pathology. Mitral valve regurgitation secondary to myocardial infarction was considered acute if the interval between myocardial infarction and operation was less than 4 weeks and chronic if the interval was greater [3,12].

Patients were considered to undergo salvage operation when brought to the operating room (OR) under cardiopulmonary resuscitation, emergency operation when brought to the OR directly from the cath lab or intensive care unit because of hemodynamic instability, urgent operation when operated on during the same hospitalization as for angiography because their discharge was deemed medically unreasonable [13]. Otherwise the operation was considered elective.

The pathogenesis of mitral valve regurgitation was classified according to previous reports [14–16]. Leaflet prolapse could result from papillary muscle infarction with secondary elongation and possible chordae rupture due to altered collagen concentration [17], or by papillary muscle rupture. Restrictive leaflet motion could be due to regional papillary muscle-annular dysfunction-complex, or due to diffuse left ventricular dysfunction with generalized ventricular and mitral annular dilatation.

Left ventricular function was assessed by ventriculography or echocardiography, or both, and categorized as normal (EF > 0.5), moderately impaired (EF = 0.3 − 0.5) or severely impaired (EF < 0.3) [18].

Mortality and morbidity rates are reported according to the guidelines of the Ad Hoc Liaison Committee for Standardizing Definitions of Prosthetic Heart Valve Morbidity of The Society of Thoracic Surgeons and The American Association for Thoracic Surgery [19].

2.2. Patient characteristics

Between January 1988 and December 1998, 263 consecutive patients underwent combined mitral valve procedure and CABG at our institution. Of these, 93 (35%) had mitral regurgitation secondary to myocardial infarction and form the basis of this report.

Patients characteristics are listed in Table 1. Eighty-four patients (90%) were in NYHA functional class III or IV. A history of congestive heart failure was present in 65 patients (70%). Seven patients had undergone open heart surgery previously. Nineteen patients (20%) underwent operation while in cardiogenic shock. Three patients underwent salvage operation, whereas 16 were operated upon on an emergency basis. Eighteen other patients (19%) underwent operation on an urgent basis. Intraaortic balloon pump (IABP) was needed in 33 patients (35%) perioperatively. It was inserted in 19 patients who were preoperatively in cardiogenic shock with a systolic arterial blood pressure of <80 mm Hg (or a decrease of more than one-third of the usual values of the patient for systolic blood pressure) and urine output of less than 300 ml/24 h. In 12 other patients IABP was inserted intraoperatively because of inability to discontinue the cardiopulmonary bypass. In two patients IABP was inserted on the first postoperative day because of hemodynamic deterioration due to ongoing myocardial infarction (in one patient) and bleeding (in another patient).

Table 1
Patient characteristics (PAP, pulmonary artery pressure; PWP, pulmonary wedge pressure)

Age	63.5 ± 8.9 yr (range 39–78)
Gender (male/female)	68 (73%)/25 (27%)
NYHA (mean)	3.5 ± 0.7
II	9 (10%)
III	32 (34%)
IV	52 (56%)
Congestive heart failure history	65 (70%)
Diabetes	9 (10%)
Chronic obstructive disease	5 (5%)
Chronic renal failure/dialysis	4 (4%)
Previous open heart surgery	7 (8%)
Acute presentation	31 (33%)
Preoperative arrhythmia	49 (53%)
Shock	19 (20%)
Status	
Elective	55 (59%)
Urgent	19 (21%)
Emergency	16 (17%)
Salvage	3 (3%)
Intraaortic balloon pumping	33 (35%)
Preoperative PAP mean (mmHg)	30 ± 13
Preoperative PWP (mmHg)	18 ± 8

Thirty-one patients (33%) presented with acute myocardial infarction.

The mean EF was 0.46 ± 0.14, 20% of patients having severely depressed and 41% moderately depressed left ventricular function. Fifty-four patients had 3-vessel disease, 25 2-vessel disease and 14 1-vessel disease (with involvement of the inferior left ventricular wall). Left main disease was present in five. The majority of patients (95%) had moderate (+3) to severe (+4) mitral regurgitation.

The mechanism of mitral regurgitation is listed in Table 2. 'Regional papillary-muscle-annular dysfunction complex' relates to a lack of coaptation of leaflets caused by an inferior myocardial infarction with secondary remodeling of the left ventricular wall supporting the posteromedial papillary muscle, and annular dilatation. The remodeling process of the left ventricular wall and the papillary muscle can distort and displace the posterior papillary muscle toward the apex.

2.3. Statistical analysis

Continuous variables are expressed as mean ± 1SD. Categorical variables are presented as percentages. Univariate analysis is done with the use of χ^2 and Fisher-exact tests as appropriate. Multivariate logistic regression analysis was used to identify independent predictors of early survival.

Survival curves are presented according to the method of Kaplan and Meier. Combinations of variables related to late survival and freedom from other events are examined using Cox proportional-hazard regression. A P value <0.05 was considered to show a statistically significant difference. All calculations were done with the use of a commercially available statistical program (SPSS 9.0).

Follow-up ranged from 6 months to 12 years (mean 51 months ± 41).

Table 2
Mechanism of mitral regurgitation

	N	%
Prolapse		
Chordal avulsion and ischemic elongated papillary muscle	10	11
Papillary muscle rupture	21	23
Restrictive leaflet motion		
Regional papillary muscle-annular complex dysfunction	43	46
Global left ventricular dysfunction	19	20

3. Results

3.1. Intraoperative variables (Table 3)

Patients were operated on under moderate hypothermia (26–32°C) and antegrade or combined antegrade and retrograde blood cardioplegia. Five patients had their operation done under induced ventricular fibrillation without aortic cross-clamping. The reasons were calcified plaques of the ascending aorta and dense periaortic adhesions after previous open heart surgery. In each case the surgeon estimated it safer not to clamp the ascending aorta. The proximal bypass graft anastomoses were performed either to the previous vein grafts or to one of the internal thoracic arteries. The mean cross-clamp time was 81.2 ± 35.9 min (range 41–154), the mean cardiopulmonary bypass time 151.1 ± 61.0 min (range 74–476). CABG was done first and additional cardioplegia delivery was routed through the vein grafts. Revascularization was complete in 86 patients (92%). The LAD was revascularized in 73 patients (74% with the LIMA). The average distal anastomoses/patient was 3.43 ± 1.62.

Mitral valve replacement ($n = 63$) was done in most instances with posterior chordal preservation. Bileaflet mechanical valves were used in 59 patients and biological valves in four (Carpentier-Edwards). Acute rupture of a papillary muscle head causes prolapse of the anterior leaflet, more often than the posterior leaflet. Mitral valve repair is difficult in these patients because of the fragility of the

Table 3
Intraoperative variables ($n = 93$)

	N	Range
Cross-clamp time (min)	81.2 ± 35.9	41–154
Bypass time (min)	151.1 ± 61.0	74–476
Number of distal anastomoses/patient	3.4 ± 1.6	1–9
LAD revascularized	73 (78%)	
LIMA	54 (74%)	
Complete revascularization	86 (92%)	
Mitral valve repair	30 (32%)	
Mitral valve replacement	63 (68%)	

necrotic tissue and because of uncertainties related to left ventricular wall remodeling after acute myocardial infarction. In these patients we performed preferably mitral valve replacement with preservation of chordae tendinae of the posterior leaflet.

Mitral valve repair, when attempted, was conducted according to various techniques previously described [20–22]. Downsizing ring annuloplasty alone [23] was undertaken in six patients. Mitral valve repair was attempted in 42 patients and was successful (by reducing mitral regurgitation by at least 2 grade) in 30 patients. Chordal avulsion and ischemic elongated papillary muscle were repaired by partial resection and readaptation of the leaflet edges and by plication of the papillary muscle. Wooler annuloplasty or Carpentier ring annuloplasty was added to the repair in presence of annular dilatation. Global left ventricular dysfunction and dilatation of the mitral annulus were repaired earlier in the series by Wooler annuloplasty [20] and later on by downsizing annuloplasty [23].

Patients with regional papillary muscle-annular dysfunction were the most difficult subset of patients in this series. Ten of 12 repair failure belonged to this group. Failed techniques included Wooler annuloplasty [20], Carpentier ring annuloplasty and Alfieri stitch [21]. The reason of failure was extended scarring of the papillary muscle and the supporting left ventricular wall resulting in severe restriction of the valve by major distortion and apical displacement of the papillary muscle with excessive increase in the papillary muscle-mitral annulus distance. Subsequently, we performed mitral valve replacement in these patients unless the increase in the distance between the tip of the papillary muscle and the mitral annulus was not excessive. In the latter cases an asymmetrical posteromedial Wooler annuloplasty (for posteromedial papillary muscle scarring) was carried out.

The mean postrepair residual mitral regurgitation as controlled by intraoperative transesophageal echocardiography was 0.6 ± 1.0. Only four patients left the OR with residual grade 2 mitral regurgitation.

Associated procedures are listed in Table 4.

3.2. Outcome

There were 14 in-hospital deaths (15%). Two patients died of sepsis and multiorgan failure. Cerebrovascular insult was the cause of death in two patients. Five patients died of cardiac failure. Bleeding was the cause of death in three patients (two due to postoperative ventricular rupture). Perioperative myocardial infarction accounted for the two other in-hospital deaths.

Table 4
Associated procedures

	N
Aortic valve replacement	8
Ascending aorta patch repair	1
Left ventricular aneurysmectomy	6
Atrial septum defect closure	1
Tricuspid valve repair	1

Table 5
Univariate analysis of hospital death (PAP, pulmonary artery pressure; PWP, pulmonary wedge pressure)

	P value
Age > 70 yr	0.7
NYHA IV	0.06
Ejection fraction	0.9
Emergency	0.15
Chronic obstructive pulmonary disease	0.004
Preoperative arrhythmia	0.1
Grade 4 mitral regurgitation	0.4
PAP mean (mmHg)	0.28
PWP (mmHg)	0.33
IABP	0.002
Cardiopulmonary bypass (min)	0.01
Valve replacement/repair	0.3

Early mortality tended to be higher in patients undergoing emergency ($8/37 = 22\%$) vs. elective surgery ($6/56 = 11\%$, $P = 0.15$), as well as in patients in functional class IV (11/52 (21%) vs. 3/41 (7%), $P = 0.06$). Preoperative ejection fraction as a continuous variable, mechanism of mitral regurgitation (prolapse vs. restrictive), atrial fibrillation and age were not associated with higher early mortality (Table 5). Duration of cardiopulmonary bypass was a risk factor for early mortality (survivors vs. non-survivors 145 ± 51 vs. 186 ± 97 min, $P = 0.01$). Early mortality was not different after mitral valve replacement ($8/63 = 12.7\%$) vs. repair ($6/30 = 20\%$, $P = 0.3$). Other univariate risk factors for early mortality included perioperative use of IABP (10/33 (30%) vs. 4/60 (7%), $P = 0.002$) and chronic pulmonary obstructive disease (3/5 (60%) vs. 11/88 (13%), $P = 0.004$). In multivariate analysis perioperative use of IABP (odds ratio = 7.4 $P = 0.005$) and chronic pulmonary obstructive disease (odds ratio = 15.4, $P = 0.02$) remained independent predictors of early mortality.

Perioperative morbidity in 79 survivors included inotropic and respiratory support more than 24 h in 36 (46%) and 32 (41%) patients, respectively. Reexploration for bleeding was done in six patients (8%). Six patients had a cerebrovascular insult (8%) and four a perioperative myocardial infarction (5%). Wound debridement for mediastinitis was necessary in three patients (4%). Four patients (5%) required dialysis postoperatively.

Late death occurred in 20 patients, of which ten were directly cardiac related and five of unknown origin. Actuarial survival rates at 1, 5 and 10 years were 81, 65 and 56%, respectively (Fig. 1). Late survival was not significantly different in patients after replacement vs. repair ($P = 0.46$), but COPD (hazard ratio = 4.0, $P = 0.02$) and use of IABP (hazard ratio = 2.6, $P = 0.003$) remained as

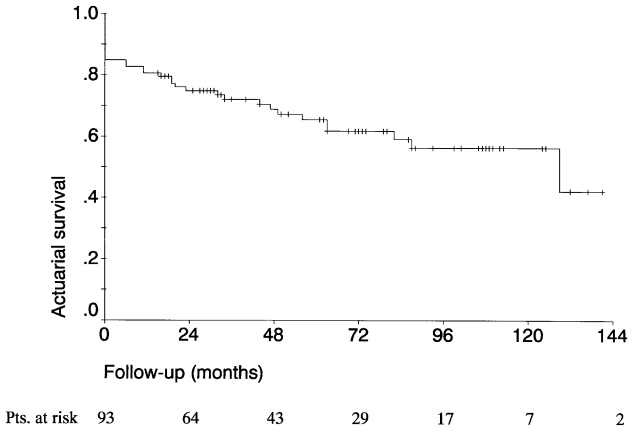

Fig. 1. Kaplan–Meier actuarial survival for the entire group.

independent predictors of decreased late survival. Fig. 2 shows the actuarial survival of patients with and without perioperative IABP. Three (two mitral valve replacements, one CABG) patients required reoperation during follow-up, resulting in a freedom from reoperation of 98, 98 and 95% at 1, 5 and 10 years, respectively. At last follow-up, all but one patient were in NYHA functional class I or II.

4. Discussion

Uncorrected moderate to severe mitral regurgitation secondary to myocardial infarction has been demonstrated to have an adverse influence on survival [1–3]. Previous reports have shown that surgical management of important mitral regurgitation secondary to myocardial infarction with CABG alone is suboptimal [3,7]. However, patients undergoing combined mitral valve procedure and CABG are at higher risk for operative death as compared to those undergoing isolated procedures [5]. In this study early mortality was 15%, which compares to 14.6% for Dion et al. [24], 9.3% for Cohn et al. [6], 9.2% for Hendren et al. [12] and 15.3% overall operative mortality for mitral valve replacement and coronary artery bypass in the STS database [5]. Early mortality tended to be higher in patients undergoing emergency surgery and in patients in preoperative functional class IV. In the STS database both factors were associated with higher operative mortality [5]. In univariate analysis the duration of cardiopulmonary bypass had a significant negative impact on early mortality. In multivariate analysis, however, the only independent risk factors for early mortality were need for perioperative IABP and COPD. Hendren et al. [12] from the Cleveland Clinic reported preoperative use of IABP to be associated with increased operative mortality. This finding underlines the negative impact of the preoperative compromised hemodynamics on early mortality. Unlike Hausmann et al. [8], we did not find preoperative ejection fraction as risk factor for early death. One explanation for this finding is that preoperative ejection fraction does not take into account hibernating myocardium which will recover after revascularization.

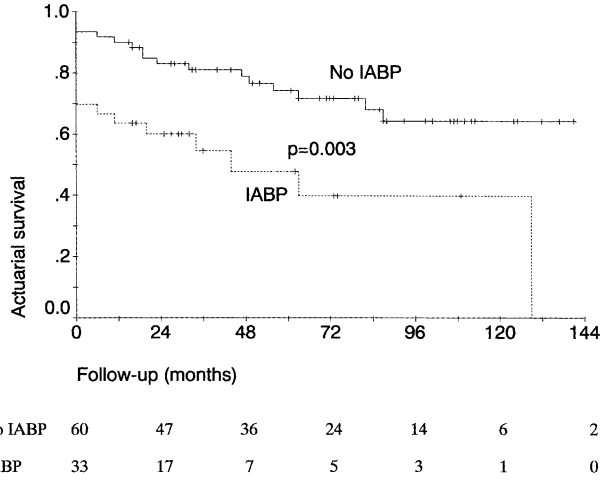

Fig. 2. Actuarial survival in patients with and without the need for preoperative IABP, showing a significantly worse outcome in those with IABP, particularly early after operation.

The 5-year survival rate in our patients is 65%, comparing with 71% for Cohn et al. [6] and 63% 3-year survival rate for the Cleveland Clinic [12].

There was no significant impact of the specific valve pathology (prolapse vs. restrictive) or mitral valve procedure (repair versus replacement) on late survival in our series. This is in accordance with the findings of other series [6,12]. Hendren et al. [12] reported an in-hospital death ratio of 11.5% for prolapse vs. 7.7% for restrictive (not significant) and could not identify significant risk factors using multivariate analyses. Univariate analysis in their study identified restrictive leaflet motion ($P = 0.02$), incomplete revascularization ($P = 0.01$), perioperative stroke ($P = 0.002$), perioperative myocardial infarction ($P = 0.01$), perioperative renal failure ($P < 0.01$), prolonged postoperative respiratory insufficiency ($P = 0.006$), preoperative intraaortic balloon pump ($P < 0.001$) as predictors of late mortality but multivariate analysis could not identify independent predictors of late mortality. In their study patients with restrictive pathology tended to fair less well after repair. Cohn et al. [6] reported an operative mortality of 9.5% for the repair group compared to 8.9% for the replacement group ($P =$ ns). There was a trend for a higher operative mortality for the functional group, and for the annular dilatation subgroup who had repair, but this was not significant. No predictors of operative mortality were identified by multivariate logistic regression analysis. In their study functional ischemic mitral regurgitation was classified as due to annular dilatation or restrictive leaflet motion. Mechanical or structural ischemic mitral regurgitation was classified as due to chordae rupture or desinsertion, and to partial or complete papillary muscle rupture. The overall 5-year survival rate was 91 ± 5% for the mitral valve replacement group and 56 ± 10% for the mitral valve repair group ($P = 0.01$). The overall 5-year survival rate for the functional group was 61 ± 10% and was not significantly different from that for

the structural group, which was 83 ± 7% ($P = 0.2$). In the study of Cohn et al. [6] multivariate logistic regression analysis identified mitral valve repair to be a significant predictor for worse long-term survival ($P = 0.014$). On further analysis, mitral valve repair was found to be a surrogate for the functional ischemic mitral regurgitation group which underwent repair ($P = 0.003$), who were directly responsible for the worse outcomes in the overall mitral valve repair group. This observation of Cohn et al. supports our finding that severe restriction with excessive increase in the papillary muscle-mitral annulus distance, and major annular dilatation are better managed by mitral valve replacement.

We recommend correction of moderate grade 3 to severe grade 4 mitral regurgitation secondary to myocardial infarction during the coronary artery bypass surgery. Although the combined mitral valve and revascularization procedure entails a significant operative risk, especially in emergency situations, the long-term benefit is substantial both in terms of survival and functional capacity. The conclusion of 'benefit in terms of functional capacity' is derived from the fact that 90% of our patients were preoperatively in functional class III or IV and postoperatively all but one survivors to follow-up in functional class I or II. The conclusion of 'benefit in terms of survival' is based on the observations of Adler et al. [2] reporting that important ischemic mitral regurgitation ($\geq +2$) left uncorrected during coronary artery bypass surgery is an independent risk factor for early and late mortality, as well as on those of Connolly et al. [3] showing that increasing severity of uncorrected ischemic mitral regurgitation during coronary artery bypass surgery correlated with increased early and late mortality. Moreover, Hickey et al. [1] showed that 5-year survival rate of patients with grades 3 and 4 ischemic mitral regurgitation treated medically was 40%. Our observed 5-year survival rate of 65% indicates a survival benefit of our surgical management compared to medical treatment in the report of Hickey et al. [1]. The limitations of our study are all those of retrospective studies. We did not directly compare the effect of bypass alone to combined mitral valve and bypass procedure in these patients. However, since grades 3 and 4 mitral regurgitation secondary to myocardial infarction are to us and other authors [1–3] indication for surgical correction, a prospective randomization of these patients to two arms with and without surgical correction of mitral regurgitation would seem unethical. The observation of Tcheng et al. [4] supports further this assertion in that they reported that patients with grades 3 and 4 ischemic mitral regurgitation had a spontaneous mortality rate of 52% at 1 year.

In conclusion, we recommend combined mitral valve procedure and myocardial revascularization, as complete as possible, for grade 3 and grade 4 mitral regurgitation secondary to myocardial infarction. The choice of mitral valve procedure should be adapted to the local anatomical findings. In our opinion acute papillary muscle rupture, severe restriction of the mitral valve by extended scarring of the papillary muscle and the supporting left ventricular wall resulting in major distortion and apical displacement of the papillary muscle with excessive increase in the papillary muscle-mitral annulus distance, and major annular dilatation are indications for mitral valve replacement. Patients with annular dilatation alone and patients with chordal avulsion and ischemic elongated papillary muscle could be satisfactorily repaired.

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Appendix. Conference discussion

Dr B. Messmer (*Aachen, Germany*): We just published in the August Journal of the Association a paper with the same topic, and we had a mortality of 19% in the ischemic and it was 6.5% in the nonischemic disease, combined mitral and coronaries. But what we have seen, I think this was a little bit surprising, that once early mortality was out for the long-term survival, it was quite similar for the patients who had mitral valve replacement or repair for ischemic combined with the coronaries as it was for degenerative or rheumatic disease combined with bypass surgery. So it seems that the big difference is early after surgery, and afterwards the patient has a very good outlook on a long-term range.

Dr Y. Zhang (*Beijing, China*): I have some questions. One is, what is your selection criteria for patients for mitral valve repair and valve replacement?

And the second, I think that your mortality is a little bit higher. If the patient is too weak to tolerate the combined procedure, how do you think just to leave the mitral valve untreated and do coronary artery bypass by a hybrid procedure, just the MIDCAB plus PTCA?

Dr Tavakoli: The first question is repair versus replacement. We tried repair in 42 patients with 30 patients successfully weaned from cardiopulmonary bypass. What does it mean successfully and what are the criteria? At this point it is very difficult to be sure what we should do with the residual mitral regurgitation. There is a paper from the Cleveland Clinic for a couple of years which shows patients with a +2 mitral residual regurgitation come back more often to reoperation late after operation. So our aim was to achieve a residual mitral regurgitation 0 to 1 + . And regarding the criteria, we think patients with acute papillary muscle rupture and patients with severe dilatation of the left ventricle and the mitral annulus are not good candidates for repair.

Would you please repeat your second question?

Dr Zhang: The patient is too weak to tolerate the combined procedure. What do you think about the hybrid procedure?

Dr Tavakoli: I think there is enough evidence in the literature that important mitral regurgitation left uncorrected affects severely the early outcome. So you have to address both problems. We have no experience with the hybrid procedure. That means valve procedure and …

Dr Zhang: No. MIDCAB plus PTCA.

Dr Tavakoli: We have no experience with hybrid myocardial revascularization in this situation.

Dr Zhang: How do you think about the size of left atrium and left ventricle and the future of the patient for symptoms is heart failure symptoms, do you think it is also included in the criteria for selection of the patients?

Dr Tavakoli: I think the impact of a history of heart failure for the surgeon in the OR was practically a question of the degree of dilatation of the left ventricle and the mitral annulus. We did in six patients a downsizing annuloplasty, as advocated by Steven Bolling, but we feel uncomfortable to do it in patients with severely dilated ventricles.

Dr H. Azar: (*Norfolk, VA, USA*): I wonder if you tried to analyze your patients in two separate groups, namely the ones who have acute mitral regurgitation in conjunction with coronary artery disease and a larger group that have chronic mitral regurgitation and see how that differs, because I think it is the second group that is going to be more in question of when to intervene and what sort of results you get.

Dr Tavakoli: I think we have not enough patients to compare acute versus chronic ischemic mitral regurgitation. This is some thing that should be done. But our impression is the preoperative hemodynamic compromise of the patient is reflected through the need for intra-aortic balloon pumping, for example, which is at least one of the determinant factors of early and late outcome.

Dr W. Binafsihi (*Jakarta, Indonesia*): I am just wondering if any of those patients could have sparring of their cords of the papillary muscle of the mitral?

Dr Tavakoli: Is the question if any patients received an artificial cord?

Dr Binafsihi: No. Could any of those patients have any potential sparring of the cords?

Dr Tavakoli: You mean during the valve replacement?

Dr Binafsihi: Yes.

Dr Tavakoli: In most instances the valve replacement was conducted with preserving at least the posterior leaflet.

Dr Messmer: I think this is very important, especially in the ischemic disease, that you keep as much suspension as you can.

Mr R. Akar (*Leicester, UK*): Mr. Azar from Virginia suggested that ischemic mitrals should be evaluated acute and chronic. I entirely agree with that, and I would like to add prolapse and restrictive ischemic mitrals has got a different prognosis as well from our series. May I ask, do you tackle grade II ischemic mitral regurgitation? You mentioned that grade III and IV is definitely an indication for surgery. What do you do for grade II?

Dr Tavakoli: We are aware of the recommendation of Dr. Dion and other colleagues, but we do not provoke patients with +2 mitral regurgitation in the operating room, but we are aware of the procedure.

Mr Akar: May I suggest that Grigone from the Mayo Clinic recently, three months ago, published his series, and same studies suggest that grade II ischemic mitral regurgitation has got an extremely high risk in long-term survival. So I think as surgeons we should think about tackling grade II ischemic mitrals.

May I ask a last question, what types of mitral repairs you performed in your repair group?

Dr Tavakoli: As I said, we used all previously described techniques by Carpentier; we used in six patients downsizing to a 28 as advocated by Steven Bolling; we used in recent patients, in five patients, accompanied by a commissuroplasty, an Alfieri stitch. So we used all possible and previously described techniques.

Mr Akar: Do you use the Alfieri stitch for ischemics?

Dr Tavakoli: Not so often. I would say in the recent patients there were only five patients who received an Alfieri stitch.